JACC Vol. 14, No. 5
November 1, 1989:1376–81

CASE REPORTS

Exercise-Induced Double (Atrial and Ventricular) Tachycardia: A Report of Three Cases

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Exercise-induced double tachycardia, i.e., the simultaneous occurrence of atrial and ventricular tachycardia, is described in three patients: one patient had coronary artery disease; the other two were young and had no apparent heart disease. One of the latter patients later died suddenly. Double tachycardia could not be initiated by programmed atrial or ventricular stimulation. In two patients atrial tachycardia always preceded ventricular tachycardia and, in one patient, ventricular tachycardia was terminated by the administration of adenosine triphosphate. Reentry does not seem to be the underlying mechanism for these arrhythmias; abnormal automaticity or triggered activity may be the mechanism.

(J Am Coll Cardiol 1989;14:1376–81)

Double tachycardia describes the simultaneous occurrence of atrial and ventricular (or junctional) tachycardia (1,2), or junctional and ventricular tachycardia (3). This uncommon tachycardia has been ascribed mainly to treatment with digitalis, generally in patients with poor left ventricular function due to coronary artery disease, and it is associated with a poor prognosis (1,2). Only a few cases of catecholamine- or exercise-induced double tachycardia, or both, have been described (4,5). We report three cases of exercise-induced double tachycardia and discuss their possible mechanism.

Case Reports

Case 1

Clinical history. A 63 year old man was admitted for an electrophysiologic study after an episode of polymorphic wide complex tachycardia was detected by Holter electrocardiographic (ECG) monitoring. Approximately 3 months before admission he underwent coronary artery bypass surgery for severe symptomatic triple vessel coronary artery disease. An ECG performed 14 days after surgery revealed recent anterior wall myocardial infarction and new complete right bundle branch block (Fig. 1). An echocardiogram showed an apical aneurysm of the left ventricle with a normal right ventricle. The patient made an uneventful recovery and was discharged without antiarrhythmic therapy. Holter ECG monitoring 3 months after surgery showed normal sinus rhythm. On one occasion, while the patient was climbing stairs, the sinus rate accelerated to 140 beats/min followed by an episode of asymptomatic, polymorphic wide complex tachycardia, at a rate of 145 to 170 beats/min that lasted for 3 min before reverting to sinus rhythm.

Electrophysiologic study. After admission, the patient underwent electrophysiologic study when he was not receiving any cardioactive drugs. The study included 1) right atrial pacing at a gradually decreasing cycle length from 600 to 270 ms; 2) programmed atrial stimulation at basic drive cycles of 600 and 400 ms using one extrastimulus; 3) programmed ventricular stimulation at the right ventricular apex and outflow tract using up to two extrastimuli delivered during sinus rhythm or at drive cycle lengths of 500 and 400 ms; and 4) right ventricular burst pacing at a gradually decreasing cycle length from 500 to 270 ms. Isoproterenol was not given because of the presence of coronary artery disease. Neither supraventricular nor ventricular tachycardia was induced during electrophysiologic study.

Effect of exercise. During the next year the patient underwent five exercise tests, always after discontinuation of cardioactive medications. Polymorphic wide complex tachycardia was always induced when the atrial rate reached a range of 145 to 190 beats/min (Fig. 2A). Esophageal ECG

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Manuscript received January 6, 1989; revised manuscript received April 19, 1989, accepted May 11, 1989.

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0735-1097/89/$3.50

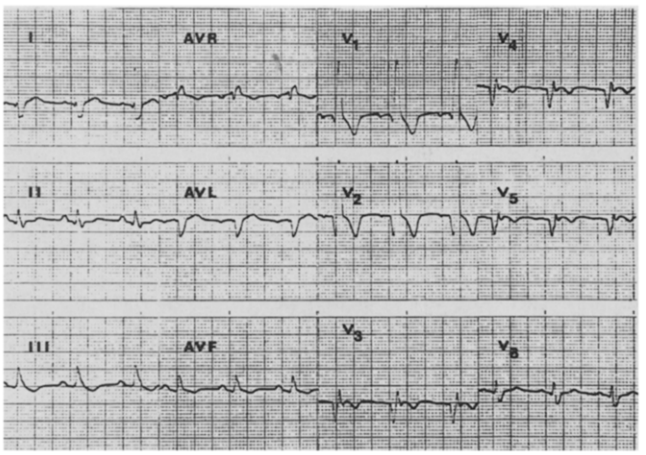

Figure 1. Patient 1. Twelve lead electrocardiogram (ECG) showing sinus rhythm, right bundle branch block and anterolateral wall myocardial infarction.

recording performed during four instances of polymorphic tachycardia revealed the simultaneous existence of two tachycardias: atrial tachycardia with a cycle length of 240 to 270 ms and ventricular tachycardia with a cycle length of 340 to 360 ms. The "polymorphic" tachycardia proved to be the result of an interplay between the two tachycardias. 1) The atrial tachycardia conducted to the ventricles with Wenckebach atrioventricular (AV) block and was associated with a right bundle branch block configuration (similar to that of the conducted sinus beats). 2) The ventricular tachycardia with a longer cycle length terminated the pause in the Wenckebach cycle, resulting in both fusion complexes and "pure" ventricular tachycardia complexes having a left bundle branch block configuration (Fig. 2B). The P waves during atrial tachycardia had a different configuration and axis from those of the P waves during sinus rhythm. After cessation of exercise, both tachycardias gradually slowed. The ventricular tachycardia was no longer manifest when the atrial tachycardia slowed and 1:1 AV conduction resumed. The atrial tachycardia then gradually slowed over 2 to 3 min and sinus rhythm supervened. During two exercise tests, episodes of monomorphic wide complex tachycardia were noted, lasting 2 to 3 min and having a left bundle branch block configuration and superior axis (Fig. 3).

Effect of adenosine during exercise. Adenosine triphosphate (10 mg) (Striadyne, Laboratories Auclair) was rapidly (<2 s) injected intravenously during induced double tachycardia immediately after cessation of exercise when atrial and ventricular cycle lengths were stable at 260 and 390 ms, respectively (Fig. 4A). Thirteen seconds later, while atrial tachycardia persisted at a cycle length of 300 ms, AV dissociation occurred, allowing the emergence of a monomorphic ventricular tachycardia having a left bundle branch block configuration at a cycle length of 390 ms (Fig. 4A and B). The cycle length of the ventricular tachycardia increased over a 4 s period to 460 ms and then abruptly terminated, whereas the atrial tachycardia continued for another 2.5 s at a cycle length of 350 ms with high degree AV block (Fig. 4C). Subsequently, 1:1 AV conduction resumed, the atrial rate gradually slowed and reverted 2 min later to sinus rhythm.

During two exercise tests performed during oral therapy with propranolol (60 mg/day), the sinus rate did not exceed

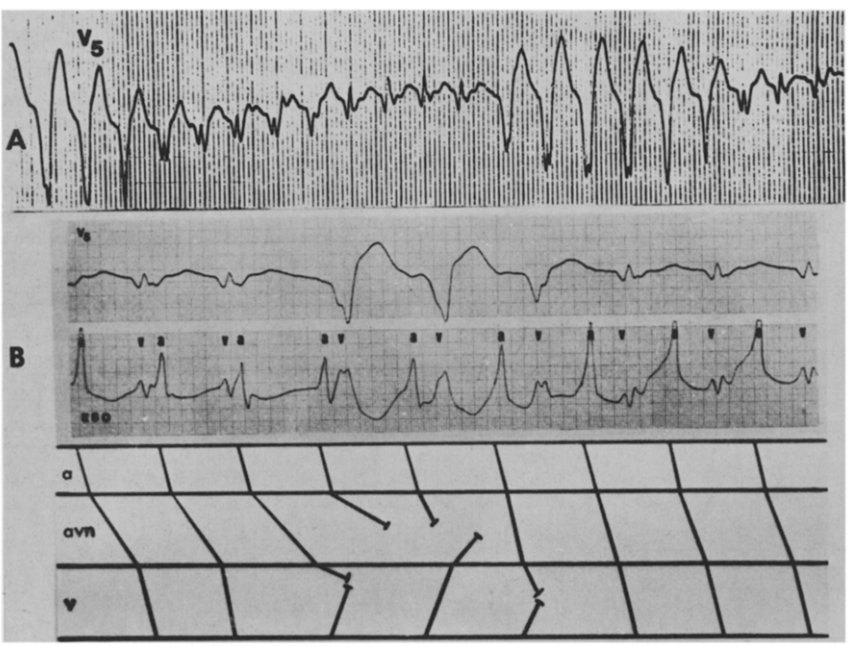

Figure 2. Patient 1. **A**, Example of polymorphic wide complex tachycardia (lead V_5) during exercise. Paper speed = 25 mm/s. **B**, Simultaneous recording of surface lead V_5 and esophageal (ESO) electrogram during polymorphic wide complex tachycardia during exercise. The ladder diagram describes the probable sequence of the Wenckebach cycle (a = atrium; avn = atrioventricular node; v = ventricle). Note that ventricular tachycardia terminates the pause in the atrioventricular Wenckebach cycle. Paper speed = 100 mm/s.

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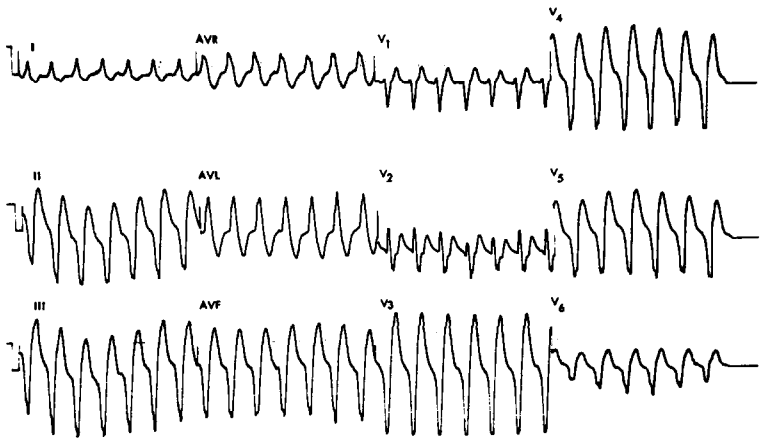

Figure 3. Patient 1. Monomorphic wide complex tachycardia at a rate of 250/min with a left bundle branch block configuration and superior axis during exercise.

140 beats/min and neither atrial tachycardia nor ventricular tachycardia was induced.

Case 2

Clinical history. This 15 year old girl was referred for evaluation because of recurrent episodes of aborted sudden death. At age 4 years she had a syncopal episode. A clinical diagnosis of seizure disorder was made and she was treated with phenobarbital. At age 9 years she had a cardiac arrest requiring cardiopulmonary resuscitation that resulted in permanent brain damage. She was referred for electrophysiologic study; prolonged sinus node recovery time and nonsustained atrial tachycardia were initiated. No ventricular tachycardia was induced by right ventricular programmed stimulation using one and two extrastimuli. Isoproterenol was not given. She was discharged on quinidine and propranolol therapy, but the latter was discontinued because she developed severe hyperactivity and bizarre behavior disorder when treated with any beta-blocking agent. Quinidine was also discontinued because of an allergic reaction. Recurrent episodes of syncope occurred. A permanent pace-

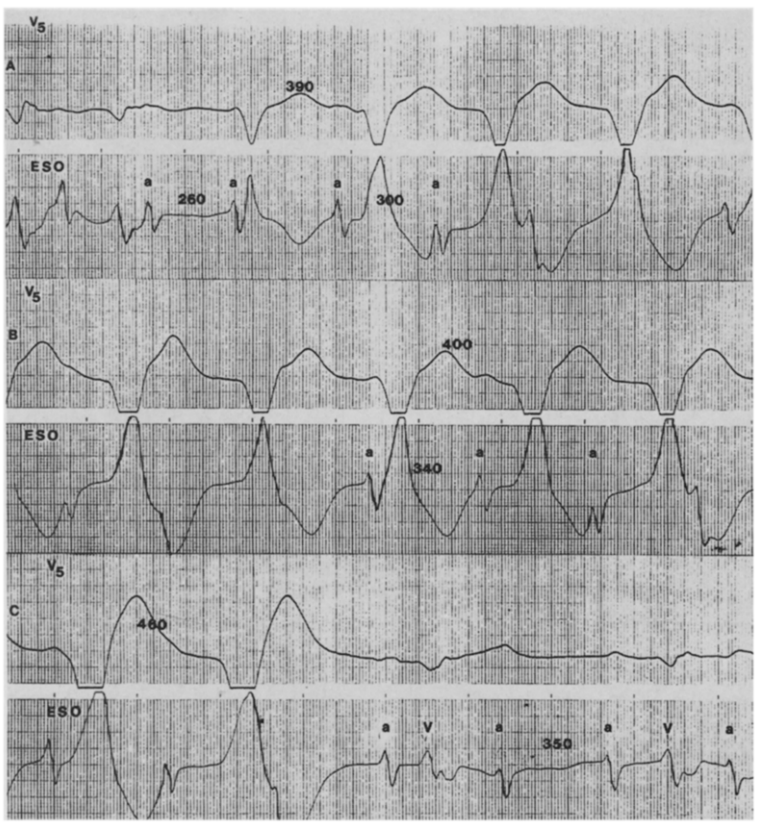

Figure 4. Patient 1. Electrocardiographic (ECG) lead V_5 and esophageal (ESO) lead recorded 13 s after administration of adenosine triphosphate during exercise-induced tachycardia. **A** and **B** are continuous and show adenosine triphosphate-induced atrioventricular (AV) dissociation and appearance of monomorphic ventricular tachycardia. Both atrial (denoted on esophageal recording) and ventricular (denoted on V_5 recordings) cycle lengths gradually increase. **C**, Further slowing and then termination of the monomorphic ventricular tachycardia 4 s after its initiation followed by continuation of the atrial tachycardia (at a cycle length of 350 ms) and high degree AV block. Note gradual widening of the QRS complex before termination of the ventricular tachycardia. Paper speed = 100 mm/s.

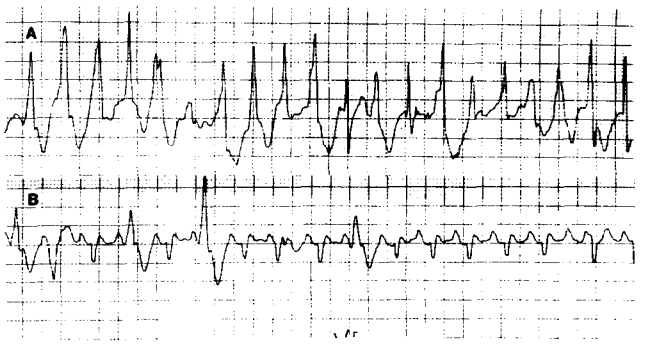

Figure 5. Patient 2. **A**, Episode of polymorphic tachycardia recorded after 3 min of exercise. **B**, Atrial flutter recorded 1.5 min into the recovery period.

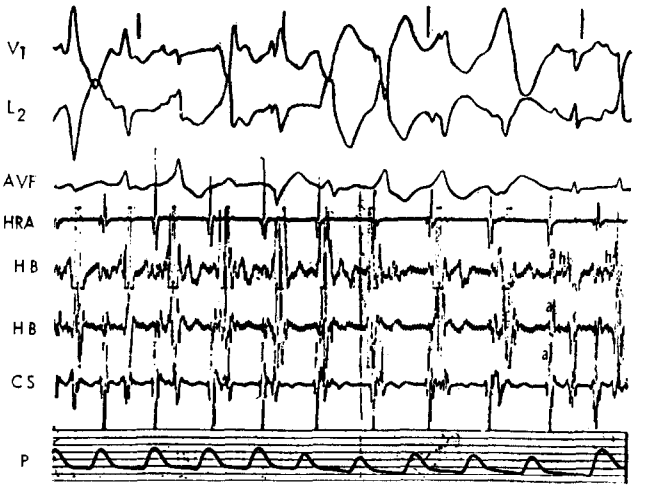

Figure 6. Patient 2. Simultaneous recordings from surface leads V_1, II and aVF, and intracardiac recordings from the high right atrium (HRA), His bundle (HB), coronary sinus (CS) and arterial pressure (P) during electrophysiologic study. After infusion of isoproterenol, both nonsustained polymorphic ventricular tachycardia and atrial tachycardia are apparent. Note that the atrial (a) deflection in the coronary sinus electrogram (CS) precedes those from the His bundle (HB) and high right atrium (HRA), thus confirming the ectopic origin of the atrial tachycardia. Conducted or fused impulses were preceded by a small His bundle deflection (h), as recorded in the last two complexes.

maker was implanted for presumed sinus node disease and she was treated with verapamil and mexiletine. Amiodarone therapy was instituted after another cardiac arrest. At the age of 16, while running, she was again resuscitated from cardiac arrest and was referred for further evaluation.

Effect of exercise. Physical examination revealed no cardiac abnormality. The ECG showed normal sinus rhythm, diffuse T wave inversion and a normal QT interval. An echocardiogram was normal. During the 1st min of treadmill exercise testing, multiple polymorphic ventricular premature complexes developed followed by repetitive runs of nonsustained polymorphic ventricular tachycardia that continued until the end of the test (3rd min) (Fig. 5A). In the recovery phase the frequency of the episodes of ventricular tachycardia decreased rapidly, and by 90 s ventricular premature complexes and atrial flutter became apparent (Fig. 5B). Sinus rhythm resumed during the 8th min of recovery.

Electrophysiologic study. The study, conducted while the patient was treated with amiodarone, revealed normal AH and HV intervals and a borderline prolongation of the corrected sinus node recovery time. Neither atrial tachycardia nor ventricular tachycardia was induced by programmed atrial or ventricular stimulation. The ventricular stimulation protocol included use of up to three extrastimuli at drive cycle lengths of 500 and 400 ms at the right ventricular apex and outflow tract. However, after isoproterenol infusion (4 μg/min), simultaneous atrial tachycardia (cycle length 380 ms) and salvos of polymorphic ventricular tachycardia (cycle length of 400 to 440 ms) appeared (Fig. 6).

Follow-up. The patient was discharged on sotalol therapy, which had to be discontinued when she developed debilitating side effects. One year later she died suddenly while riding her bicycle.

Case 3

Clinical history. An 11 year old boy was hospitalized because of episodes of palpitation and a history of syncope at age 10. Physical examination, including neurologic evaluation and electroencephalography, did not disclose any abnormality. Programmed atrial and right ventricular apical stimulation using up to two extrastimuli did not induce either supraventricular tachycardia or ventricular tachycardia. Isoproterenol was not given. The ECG showed normal sinus rhythm and inverted T waves in leads aVL and V_1 to V_3 (juvenile pattern). An echocardiogram was normal.

Effect of exercise. Over the next 6 months several exercise treadmill tests were performed, all showing simultaneous atrial tachycardia and ventricular tachycardia (cycle length 320 to 380 ms and 300 to 360 ms, respectively) (Fig. 7). Atrial tachycardia always appeared first with negative P waves in lead II, III, aVF and V_2 to V_6, followed shortly by simultaneous ventricular tachycardia, which had a left bundle branch block configuration and superior frontal plane QRS axis with fusion and capture beats resulting in a polymorphic pattern (Fig. 7). The double tachycardia then continued for 1 to 2 min into the recovery phase. The atrial tachycardia always terminated first followed shortly by an abrupt termination of the ventricular tachycardia. On several occasions either nonsustained or sustained ventricular tachycardia succeeded episodes of nonsustained atrial tachycardia during the recovery period of an exercise test (Fig. 8). In these studies (which were performed on different days) a direct relation could be found between the cycle

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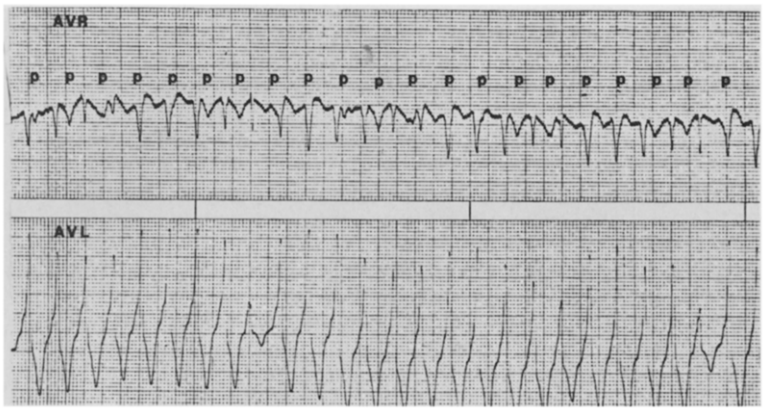


Figure 7. Patient 3. Simultaneous recording of leads aVR and aVL during exercise shows simultaneous atrial (P) and ventricular tachycardias with an apparent polymorphic configuration.

lengths of the atrial tachycardia and the ensuing ventricular tachycardia (Fig. 8).

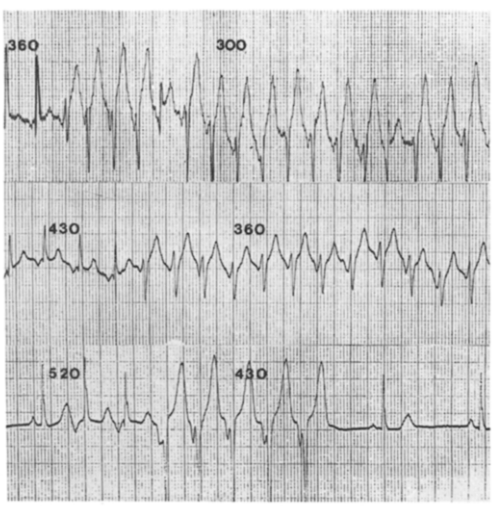
Follow-up. Therapeutic trials with quinidine and amiodarone did not prevent induction of the double tachycardia by exercise. The patient was discharged without antiarrhythmic treatment and has remained asymptomatic during a follow-up period of 8 years.

Discussion

Diagnosis of double tachycardia. Double tachycardia is a relatively uncommon type of arrhythmia (1–3). Only a few cases of catecholamine- or exercise-related double tachycardia have been reported (4,5), possibly because of difficulty in diagnosis. In our patients, atrial activity during double tachycardia was not evident on the surface ECG in Patient 1 and 2, and the P waves were evident in only one lead (aVR) in Patient 3 (Fig. 7). Motte et al. (6) have emphasized the importance of esophageal recordings in double tachycardia. The findings in Patient 1 show that esophageal recording during exercise-induced double tachycardia is feasible and may be essential for correct diagnosis.

The configuration of the double tachycardia also presented interesting findings. In all three patients, capture and fusion beats occurred. In Patient 1 and 3, the interplay between atrial capture and ventricular tachycardia could have been interpreted as polymorphic ventricular tachycardia. There have been several previous reports (7–9) of exercise-induced polymorphic ventricular tachycardia; it is conceivable that some of these arrhythmias may have been double tachycardia. The recording of atrial activity during the polymorphic tachycardia may provide an important clue to uncovering of the underlying mechanism.

Figure 8. Patient 3. Three examples of initiation of ventricular tachycardia by atrial premature beats during recovery period of exercise test. In all strips ectopic atrial beats are followed by the emergence of ventricular tachycardia. Note the direct relation between the cycle lengths of the atrial and ventricular rhythms. The numbers denote cycle lengths in milliseconds.



Pathophysiology of exercise-induced double tachycardia. The pathophysiology is unknown. Two of our patients (Cases 2 and 3) were young and had no apparent heart disease. The third patient (Case 1) had coronary artery disease, and the ventricular tachycardia was documented for the first time during routine ambulatory ECG (Holter) monitoring after an acute myocardial infarction. However, because the arrhythmia was seldom associated with any symptoms, it could have occurred undetected for years. It is unlikely that it was initiated by acute myocardial ischemia because no clinical or ECG evidence of ischemia preceded the emergence of the double tachycardia during the exercise tests.

In two patients (Cases 1 and 3), atrial tachycardia or ectopic atrial beats always preceded the ventricular tachycardia, suggesting initiation of ventricular tachycardia by supraventricular beats. The induction of ventricular tachycardia by atrial beats (either spontaneously or by atrial pacing) has been previously reported (6,10–13). Interestingly, ventricular tachycardia (and double tachycardia) were

not initiated during electrophysiologic study in these two patients. However, atrial tachycardia or atrial beats preceding the double tachycardia always occurred during or immediately after exercise when it is presumed that plasma catecholamine levels were high. Conceivably, catecholamine administration during the electrophysiologic study might have facilitated the induction of tachycardia in these patients. In fact, double tachycardia in Patient 2 occurred during the electrophysiologic study only after infusion of isoproterenol.

Electrophysiologic mechanism. The mechanism of the exercise-induced double tachycardia in our patients remains speculative. Several characteristics of the ventricular tachycardia in Patient 1 and 3 seem incompatible with reentry: 1) ventricular tachycardia could not be induced by programmed ventricular stimulation; 2) it was repetitively initiated by exercise, but without a preceding ectopic complex; and 3) ventricular tachycardia was terminated by adenosine triphosphate administration in Patient 1 (14). Several phenomena implicate triggered activity due to rate-dependent augmentation of delayed afterdepolarization as the underlying mechanism of the double tachycardia. These include 1) initiation of the double tachycardia by either sinus tachycardia or atrial tachycardia in Patient 1 and 3 (12,15); 2) the direct relation between the cycle length of the atrial beats initiating the double tachycardia and the rate of the following ventricular tachycardia in Patient 3 (16–18); and 3) termination of ventricular tachycardia by adenosine triphosphate in Patient 1 (16,19). Interestingly, the atrial tachycardia rate slowed in Patient 1 after the administration of adenosine triphosphate (Fig. 4C). However, these observations cannot definitely distinguish triggered activity from abnormal automaticity (20–22).

Summary. Two important features of our three cases deserve emphasis. 1) Exercise-induced double tachycardia may simulate polymorphic ventricular tachycardia and esophageal ECG studies may help in differentiation. 2) Abnormal automaticity or triggered foci may reside in both the atrium and the ventricle and produce double tachycardias. Beta-blocker therapy was effective in preventing exercise-induced tachycardia in one case but produced intolerable side effects in a second case.

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